Case Report

jfms OPEN REPORTS

Journal of Feline Medicine and Surgery Open Reports
1–7

DOI: 10.1177/2055116919861248
journals.sagepub.com/home/jfmsopenreports

This paper was handled and processed by the European Editorial Office (ISFM) for publication in *JFMS Open Reports*

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Hyperplastic and fibrosing gastropathy resembling Ménétrier disease in a cat

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Abstract

Case summary A 3.5-year-old domestic shorthair cat presented with a 6 month history of weight loss and polyphagia. Clinical examination revealed a markedly reduced body condition score (2/9) and a quiet demeanour. Laboratory abnormalities comprised a mild non-regenerative anaemia, stress leukogram, hypoproteinaemia due to hypoalbuminaemia, azotaemia, hypokalaemia, total hypocalcaemia and sub-maximally concentrated urine (specific gravity 1.020). Abdominal ultrasonography revealed marked thickening of the gastric mucosa within the fundus, body and pylorus; the most dorsal portion of the fundus was spared. The thickened mucosa contained multiple small, anechoic cyst-like structures. The gastric submucosa, muscularis and serosa appeared normal. Histopathology, performed on a full-thickness gastric biopsy, revealed mucosal hypertrophy and markedly dilated gastric glands in areas; not all gastric glands were affected, with some appearing normal or atrophic. Focal interstitial fibrosis was present in some areas. The findings of hypoproteinaemia, gastric ultrasonographic changes and histopathology results share several similarities to those reported with Ménétrier disease.

Relevance and novel information Ménétrier disease is a rare condition of the stomach in humans. A similar condition, giant hypertrophic gastritis (or Ménétrier-like disease), has also been described rarely in dogs. To our knowledge, Ménétrier-like disease has not been previously described cats. This case shares features of Ménétrier-like disease, raising the suspicion of a similar aetiopathogenesis.

Keywords: Gastropathy; giant hypertrophic gastritis; stomach; ultrasound

Accepted: 10 June 2019

Introduction

Ménétrier disease, also known as giant hypertrophic gastritis or hypoproteinaemic hypertrophic gastropathy, is a rare idiopathic condition of the stomach in people. It was first described by the French pathologist Pierre Ménétrier in 1888.[1] Diffuse thickening of the gastric wall (frequently sparing the antrum) and giant rugal folds are typically seen on abdominal imaging (radiography, CT and ultrasonography) and gastroscopic examination. Diagnosis of Ménétrier disease requires full-thickness biopsy of the abnormal gastric wall. Histopathological features of the disease are marked expansion of the surface mucus cells (foveolar hyperplasia), cystic dilatation of the glandular portion of the gastric mucosa, reduced numbers of parietal and chief cells (glandular or oxyntic atrophy) and the absence of significant inflammatory infiltrate. A link to gastric cancer has been recognised in people.[1–3]

Giant hypertrophic gastritis, or Ménétrier-like disease, has been reported in an 11-year-old male Old

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English Sheepdog,[4] a 4-year-old male Jack Russell Terrier[5] and a 7-year-old Boxer.[6] Ménétrier-like disease has also been reported in association with gastric carcinoma in a West Highland White Terrier[7] and gastric adenocarcinoma in three Cairn Terrier littermates.[8] A breed-specific form of gastric hypertrophy has been described in the Basenji; however, in contrast to Ménétrier disease, histopathological examination identified only infrequent glandular cysts and concurrent lymphoplasmacytic gastroenteritis.[9]

Case description

A 3.5-year-old male neutered domestic shorthair cat was referred for investigation of a 6 month history of progressive weight loss (40% from 2 years prior) and marked polyphagia. Hunting and eating of prey, and scavenging of human food (meat, fruit, vegetables), including from refuse and directly from the owner, was a feature. Vomiting was infrequently reported, comprising foam or liquid. Faeces were reported to be normal, and there was no history of flatulence or borborygmus. Other than altered appetite, the cat's behaviour was considered to be normal. The cat had limited outdoor access and was fed a complete commercial dry diet (Iams Adult with Chicken; Procter & Gamble). The cat was fully vaccinated and had received anthelmintic treatment (unknown product) within the 6 month period prior to referral.

The cat had a prior history of osteopenia resulting in femoral and vertebral folding fractures at 6 months of age. The cat was managed with a non-steroidal anti-inflammatory (meloxicam 0.05 mg/kg q24h PO [Metacam 0.5 mg/ml oral suspension for cats; Boehringer Ingelheim]; discontinued after 3–4 days once comfortable), exercise restriction, and calcium and vitamin D3 supplementation (elemental calcium 120 mg/kg, cholecalciferol 2 µg/kg [Pet-Cal; Pfizer]; discontinued after 6 months). Renal secondary hyperparathyroidism was excluded as a cause of the osteopenia; however, dietary information was not available at that time and full characterisation of the underlying aetiology was not performed. No further skeletal problems were reported.

On presentation the cat was quiet, alert and responsive. General clinical examination revealed markedly reduced body condition (score 2/9; weight 1.83 kg) with poor muscle mass, and palpably small kidneys. The remainder of physical examination, including assessment of the mucous membranes, eyes, skin and coat, was unremarkable. On abdominal palpation no discomfort was noted and the intestines were unremarkable.

Routine haematology and serum biochemistry revealed: a mild normochromic–normocytic anaemia; a mild mature neutropenia and eosinopenia; mild azotaemia; moderate hypoproteinaemia due to mild hypoalbuminaemia; marked hypokalaemia; and a mild total hypocalcaemia with normal ionised calcium levels (Tables 1 and 2). Serum folate and cobalamin analysis revealed a normal folate level and a marked hypocobalaminaemia. Coagulation assessment was unremarkable (Table 3). Urinalysis revealed a urine specific gravity of 1.020, a mild proteinuria, and small numbers of erythrocytes, scant leukocytes and bacteria noted on sediment examination; however, urine bacterial culture was negative (Table 4).

Abdominal ultrasonography (Siemens Acuson S2000 using a 14L5 linear transducer) revealed marked changes to the gastric fundus (ventrally), body and pylorus (Figure 1). The dorsal part of the fundus appeared normal. The mucosa was markedly thickened (up to 14 mm; normal overall gastric wall thickness

Table 1 Haematology

Parameter	Result	Reference interval
Haemoglobin (g/dl)	7.78*	8.00–15.00
Haematocrit (%)	24.2*	25.0–45.0
RBC count ($\times 10^{12}$/l)	4.90*	5.50–10.00
Mean cell volume (fl)	49.4	40.0–55.0
Mean cell haemoglobin (pg)	15.9	12.5–17.0
Mean cell haemoglobin concentration (g/dl)	32.1	30.0–35.0
Platelets ($\times 10^9$/l)	340	200–700
WBC count ($\times 10^9$/l)	20.60*	4.90–19.00
Neutrophils ($\times 10^9$/l)	15.66*	2.40–12.50
Lymphocytes ($\times 10^9$/l)	4.33	1.40–6.00
Monocytes ($\times 10^9$/l)	0.62	0.10–0.70
Eosinophils ($\times 10^9$/l)	0.00*	0.10–1.60
Basophils ($\times 10^9$/l)	0.00	0.00–0.10

Smear examination: normocytic, normochromic red blood cells, mild anisocytosis
*Indicates a result outside the reference interval
RBC = red blood cell; WBC = white blood cell

Table 2 Serum biochemistry

Parameter	Result	Reference interval
Urea (mmol/l)	11.6*	6.5–10.5
Creatinine (μmol/l)	153	133–175
Total protein (g/l)	56.6*	77.0–91.0
Albumin (g/l)	22.9*	24.0–35.0
Globulin (g/l)	33.7	21.0–51.0
Albumin/globulin ratio	0.68	0.40–1.30
Alanine aminotransferase (IU/l)	52*	15–45
Alkaline phosphatase (IU/l)	14	15–60
Total bilirubin (μmol/l)	3.1	0.0–10.0
Sodium (mmol/l)	148.7*	149.0–157.0
Potassium (mmol/l)	2.64*	4.00–5.00
Chloride (mmol/l)	116	115–130
Calcium (mmol/l)	2.25*	2.30–2.50
Ionised calcium (mmol/l)	1.22	1.10–1.40
Phosphate (mmol/l)	1.42	0.95–1.55
Cholesterol (mmol/l)	3.9	2.3–5.3
Folate (ng/l)	16.9	7.0–21.0
Cobalamin (ng/l)	<150*	>183
TLI (μg/l)	24.4	12–82
Thyroxine (nmol/l)	20	19–62

*Indicates a result outside the reference interval
TLI= trypsin-like immunoreactivity

Table 3 Coagulation panel

	Result	Reference interval
PT control (s)	8.7	7.0–9.0
PT (s)	8.3	7.0–9.0
APTT control (s)	17.3	14.0–18.0
APTT (s)	16.0	14.0–18.0

PT = prothrombin time; APTT = activated partial thromboplastin time

reported to be 1.7–3.6 mm)[10] and contained multiple 2–3 mm diameter, round, anechoic cyst-like structures. The gastric submucosa, muscularis and serosa were normal in appearance. The stomach was devoid of content, with a collapsed lumen and no evidence of rugal folds. Both kidneys were small (left 27 mm; right 31 mm [normal 30–45 mm]),[11] slightly irregular in outline and contained multiple small calculi. The remainder of the abdomen was unremarkable.

Following correction of the hypokalaemia with potassium-supplemented intravenous fluid therapy (compound sodium lactate, with additional potassium 60 mmol/l; 4 ml/kg/h), an exploratory coeliotomy was performed. The gastric wall was visibly, and palpably, extremely thickened and irregular (Figure 2). No gross lesions affecting the liver, spleen, pancreas, intestines, kidneys, lymph nodes or omentum were observed. Full-thickness gastric, duodenal, jejunal and ileal biopsies were collected for histopathological analysis. The small intestine was biopsied with a 3 mm punch, closing with a single layer of PDS sutures. An incisional biopsy of the stomach was performed, removing a single 1 cm^2 section, closing with a double layer of polysorb. Both the small intestinal and stomach biopsy sites were omentalised.

During recovery (1 h postsurgical closure) the patient developed severe hypotension, hypothermia (34°C) and severe anaemia, which was refractory to crystalloid and colloid fluid boluses, ephedrine, norepinephrine and type-matched whole blood transfusion. The cat was subsequently euthanased owing to further decline in its condition. The owner declined a post-mortem examination.

The predominant histopathological finding was of gastric mucosal hypertrophy (Figure 3), with variable, often marked, dilatation of some gastric glands affecting various levels of the mucosa. These dilated gastric glands were lined by poorly differentiated cuboidal cells; other gastric glands were normal or atrophic. In one area of marked glandular change there was mild interstitial

Table 4 Urine biochemistry (collection by cystocentesis)

Parameter	Result	Reference interval
pH	6.3	
Blood	Positive ++	
Protein:creatinine ratio	0.77	<0.50
Glucose	Negative	
Ketones	Negative	
Protein (mg/dl)	78.8	
Creatinine (mmol/l)	9.0	
Specific gravity	1.020	

Sediment examination: scant fat droplets, red blood cells (+), bacteria (++), scant white blood cells, scant epithelial cells

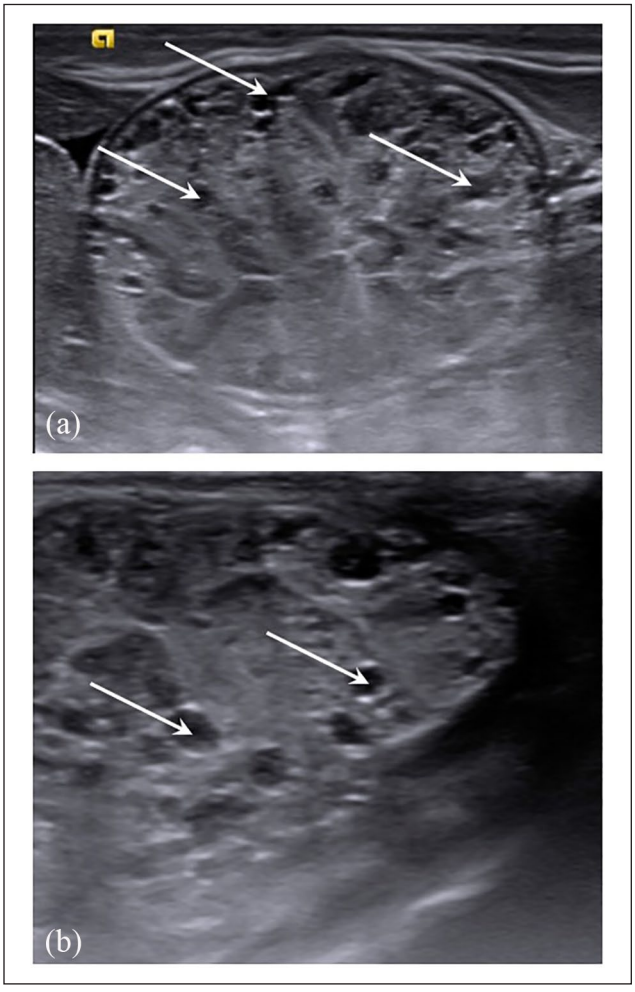

Figure 1 Ultrasonographic images of the stomach in the (a) axial plane through the body of the stomach, and (b) longitudinal plane. Note the marked thickening of the mucosa (up to 14 mm; normal overall gastric wall thickness 1.7–3.6 mm),[9] which contains multiple small (2–3 mm diameter), rounded anechoic cyst-like structures (selection indicated by arrows). The normal layered appearance of the muscularis and serosa is present. The stomach is empty

Figure 2 Intraoperative photograph showing the irregular, cerebriform-like appearance of the gastric serosal surface. *Courtesy of Ivan Doran*

fibrosis and inflammation, with infiltration by small numbers of lymphocytes and plasma cells. In other areas of mild interstitial inflammation there was atrophy or loss of glandular tissue. Occasional small clusters of lymphocytes and plasma cells were present in the superficial lamina propria adjacent to relatively normal mucosal tissue. The superficial gastric pits, muscularis and serosa were normal. There was no evidence of neoplasia. Small intestinal histopathology was unremarkable.

Discussion

This report describes the case of a young adult cat, presenting with clinical features consistent with

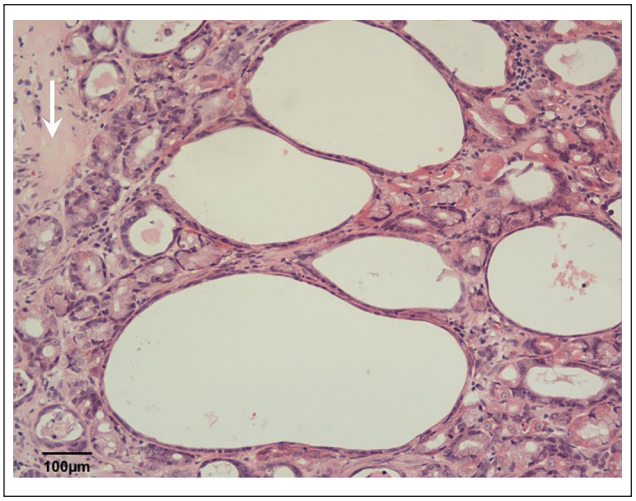

Figure 3 Histological section of gastric mucosa showing variably dilated gastric glands, lined by poorly differentiated cuboidal cells. There is also some interstitial fibrosis (arrow). Haematoxylin and eosin

gastrointestinal dysfunction (weight loss, polyphagia, hypoalbuminaemia, hypocobalaminaemia), and ultrasonographic and histopathological changes consistent with giant hypertrophic gastritis ('Ménétrier-like disease'). The precise aetiology of the gastric mucosal changes in this patient remains unclear. Reports of similar gastric changes in the veterinary literature are few and difficult to identify; only a small number of canine cases have been described, and none in cats.

The mild anaemia was considered most consistent with anaemia of inflammatory disease. The neutrophilia and eosinopenia were considered most consistent with a 'stress' leukogram. Considering the urine specific gravity of 1.020, the mild azotaemia was considered most likely renal in origin, with the poor muscle mass contributing to the creatinine remaining within the reference interval (measurement of symmetric dimethylarginine was not possible, as this assay was not commercially available at that time). The moderate hypoproteinaemia, with the mild hypoalbuminaemia, was considered most likely secondary to gastrointestinal dysfunction (either as a result of malassimilation or malabsorption). With a partial contribution of urinary protein loss, a negative acute-phase protein response could not be excluded. The mild total hypocalcaemia with normal ionised calcium levels was attributed to reduced fraction of calcium bound to albumin. The marked hypokalaemia was considered most likely secondary to ongoing renal losses, given the continued oral intake of food, absence of diarrhoea and absence of third-spacing. As the cat had been fed a complete commercial diet and exocrine pancreatic insufficiency (EPI) had been ruled out, the marked hypocobalaminaemia was attributed to gastrointestinal dysfunction. In the absence of significantly altered hepatic enzyme activity, normal cholesterol, and unremarkable ultrasonographic and gross appearance of the liver, hepatic dysfunction was not considered likely and dynamic bile acids were not assessed. Hyperthyroidism, as a cause of the polyphagia, was excluded, as there was a low-to-normal thyroxine level.

Ultrasonographic findings (small, irregular kidneys, containing multiple small calculi) were consistent with chronic kidney disease (minimum International Renal Interest Society stage 1), which further supported the low urine specific gravity and mild azotaemia being renal in origin. The inciting cause of the kidney changes was unknown. The early chronic kidney disease was considered unlikely to have been related to the gastric wall changes, as the elevated urea was only mild, and histopathological changes were not consistent with a uraemic gastropathy. Although the urinalysis was suggestive of an active urinary sediment, bacterial culture was negative (Table 4). Possibilities included failure to culture, presence of uncultivatable organisms or artefact. Owing to the close proximity of the hospital to the diagnostic laboratory, urine would have been rapidly processed for culture, which would minimise the likelihood of failure to culture cultivatable organisms. There was no history of antibiotic administration that could also have contributed to a failure to culture. Sediment examination revealed only scant white blood cells alongside red blood cells, such that some of these could be as a result of haemorrhage. The cat exhibited no clinical signs consistent with cystitis.

This cat was euthanased as a result of complications following gastrointestinal biopsy. No site of haemorrhage was identifiable on focused ultrasonographic examination of thoracic or abdominal cavities, and therefore intraluminal haemorrhage was suspected.

Hypertrophic-to-fibrosing gastritis has been reported as an inconsistent histopathological feature in cats infected with the small nematode *Ollulanus tricuspis*;[12–14] however, concurrent gastric pathology in reported cases (trichobezoar; adenocarcinoma; *Helicobacter felis* infection) limits conclusions as to the significance of this parasite. In one study, endoscopic examination was unremarkable in cats subsequently found to be infected with *O tricuspis* on histology, with additional findings of mild, chronic gastritis in only 2/4 parasitised cats.[12] An earlier study assessing the prevalence of *O tricuspis* found 13% of cats submitted for post-mortem examination to be infected, with none showing gross changes.[15] Inconsistently reported clinical signs in cats with *O tricuspis* infection include vomiting, anorexia and weight loss.[12–14] Eosinophilia was not a feature of previously described cases of *O tricuspis* infection, and serum albumin levels were not reported.[12,13] In the present case no evidence of parasitic infection was identified,

although biopsy histology is reported to be an insensitive technique for *O tricuspis* detection,[12] with stomach scrapings and gastric lavage being preferred methods.[15] Administration of prophylactic anthelmintics may also have reduced the sensitivity of detection of parasitism. *Helicobacter* species infections have been associated with mild chronic gastritis in cats,[16] and with gastric adenocarcinoma in several species, including humans.[17]

A further differential for gastric wall thickening, considered prior to biopsy, would be neoplasia. B-cell lymphoma is the most common type of feline gastric tumour;[18] however, this and other types of tumour, such as gastrointestinal stromal tumours and carcinomas, would be more likely to present as discrete masses with loss of mural layering. Gastric carcinoma has been suspected to be a sequela of Ménétrier disease in people, and Ménétrier-like disease in dogs.[7,8,19] No histological evidence of neoplasia was identified in the cat described here, nor were any spiral bacteria detected; however, as only a single gastric site was biopsied this cannot be fully excluded.

Ménétrier disease in humans is a rare idiopathic gastric disorder characterised by cystic mucosal hyperplasia. It most frequently affects men between the age of 30 and 60 years.[3] Common symptoms include abdominal pain, nausea, vomiting and oedema of the peripheral tissues.[2,20] A Ménétrier-like disease has been rarely reported in typically older dogs, with the main clinical signs being chronic vomiting and weight loss.[4,6–9] Unusually, the cat presented here was a young adult; however, a transient form of Ménétrier disease has rarely been reported in children,[21,22] and Ménétrier-like disease has been described in young adult dogs.[9] Progressive weight loss and polyphagia were features of this cat's clinical signs, which are more typical of a malassimilation or malabsorptive state; surprisingly, vomiting was not a significant feature.

Ménétrier disease in humans is often accompanied by hypoalbuminaemia and hypochlorhydria, secondary to protein leakage across the gastric mucosa and reduced numbers of parietal cells, respectively.[2,23] Hypoalbuminaemia has also been reported in dogs with Ménétrier-like disease.[4,6,7] Mild hypoalbuminaemia and moderate hypoproteinaemia were present in the cat described here. The hypokalaemia, present in the cat reported here, has not been described in Ménétrier disease in humans or Ménétrier-like disease in dogs. It is most likely related to increased renal losses due to chronic kidney disease.

The ultrasonographic appearance of Ménétrier disease in humans is associated with grossly thickened and lobulated gastric folds.[21,24] Abdominal ultrasonography has also proved a useful non-invasive tool in the diagnosis of Ménétrier-like disease in the dog,[4,7,8] where diffuse gastric wall thickening containing multiple cystic-like lesions, comparable to the ultrasonography findings in the cat presented here, have been noted.

Definitive diagnosis of Ménétrier disease in humans requires histopathological demonstration of cystic glandular dilatation and glandular atrophy;[25] very similar histopathological findings were reported in the cases of canine Ménétrier-like disease and in this cat.[4,6–9] However, fibrosis between the affected glands is not described as a feature of Ménétrier disease in humans, but is a described in some cases of canine Ménétrier-like disease.[8]

Heightened epidermal growth factor receptor (EGFR) activity and increased expression of one of its ligands, transforming growth factor (TGF)-α, have been implicated in the pathogenesis of Ménétrier disease in humans.[26,27] The pathomechanisms resulting in increased TGF-α expression associated with Ménétrier disease have not been fully elucidated. In a small subset of patients there is an association between Ménétrier disease and the presence of *Helicobacter pylori*, with disease regression following treatment of the infection.[27] Recent attempts to manage severe cases of Ménétrier disease with cetuximab, a monoclonal antibody that binds to and blocks the activity of EGFR, have shown promising results.[1,3] Lanreotide, and other somatostatin analogues, have also been described in the successful management of Ménétrier disease, and are thought to inhibit EGFR activity by both direct and indirect means.[28]

To support a diagnosis of Ménétrier-like disease, cytological examination of the gastric fluid, and histopathological examination of the liver and pancreas, could have excluded other differentials for the polyphagia with weight loss such as gastric parasitism (eg, *O tricuspis* infection), hepatopathy, pancreatitis or pancreatic atrophy. In humans with suspected Ménétrier disease, measurement of gastric fluid pH and chloride concentrations are considered. As only one biopsy was collected from each segment of small intestine, focal disease affecting an area not biopsied cannot be excluded. No clear link could be established between the reported developmental osteopenia and gastric changes in this case. No orthopaedic concerns were described by the owners at presentation.

Hypocobalaminaemia is not reported as a complication of Ménétrier disease in people or Ménétrier-like disease in dogs; however, it is not apparent from medical and veterinary case reports that serum cobalamin concentrations were measured in those cases. The hypocobalaminaemia in this case could have been secondary to hypochlorhydria resulting in malassimilation, having excluded EPI, enteropathy and hepatopathy (as far as possible on the basis of diagnostic tests performed), and the absence of diarrhoea.

Conclusions

We present a novel case of hyperplastic and fibrosing gastropathy in a cat, associated with an unusual ultrasonographic appearance of the stomach, which, to our

knowledge, has not been previously reported in this species. The marked gastric wall thickening, hypoproteinaemia with hypoalbuminaemia, and cystic dilatation of the mucosal glands resemble some of the features of Ménétrier disease in humans and Ménétrier-like disease in dogs. A gastropathy characterised by mucosal gland hyperplasia and fibrosis should be considered in cats with marked thickening and a cystic appearance of the gastric mucosa identified on abdominal ultrasonography.

Acknowledgements The authors would like to acknowledge Ivan Doran for the picture provided in Figure 2, and Professor Ed Hall for his assistance with this case.

Conflict of interest The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding The authors received no financial support for the research, authorship, and/or publication of this article.

Ethical approval This work involved the use of a client-owned animal only, and followed internationally recognised high standards ('best practice') of individual veterinary clinical patient care. Ethical approval from a committee was not therefore needed.

Informed consent Written informed consent was obtained from the owner of the cat described in this work for the procedures undertaken.

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